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Cloth Ballistic Vest Alters Response to Blast

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Ballistic wounds have been and will remain the principal cause of casualties
in combat. Cloth ballistic vests (CBV) play an important role in limiting
critical wounds from fragments and small-arms fire. There is an increased risk
of primary blast injury on the modern battlefield. In a previous study,
volunteers were exposed to short-duration blast waves of low peak pressure
(18.6 ± 0.8 kPa). Pressure measurements made in the distal esophagus as an
estimate of intrathoracic pressure (ITP) were significantly higher ($p < 0.05$)
when the standard U.S. Army ballistic jacket was worn (8.7 ± 1.2 kPa) than
when fatigues alone were worn (7.4 ± 0.7 kPa). In this study 58 sheep were
exposed to nominal blast levels of 115, 230, 295, and 420 kPa peak pressure
in groups of 12, 18, 16, and 12, respectively. Half of each group was fitted
with a CBV. Lung weight index (LWI), lung weight expressed as a percentage
of body weight, was used as a measure of blast injury. Use of the CBV was
associated with a significant increase in LWI ($p < 0.05$) which averaged 21%
for the two middle exposure groups. At the 420 kPa level, two of six non-CBV
animals died as opposed to five of six animals wearing the CBV. Intrathoracic
pressure was generally higher in the CBV group. Likely mechanisms of injury
enhancement include an increase in target surface area and an alteration of
the effective loading function on the thorax. This information may be useful in
the triage and treatment of casualties exposed to intense blast environments.

Explosions are the coinage of modern warfare. Damage is done to personnel or material by the direct cussive action of the blast wave (primary effect) or by the energizing of fragments or environmental debris as penetrating missiles (secondary effects). Tertiary blast effects are a result of the body being translated by a very strong explosion as with a nuclear burst. All mention of blast in this paper infers a primary effect unless otherwise stated.

The overwhelming risk to a soldier on a conventional battlefield has been, and will remain, wounding from ballistic particles. These may come from small-arms fire or from exploding ordnance. Blast has not been recognized as a major cause of combat casualties. However, submersion blast injury has been reported frequently, including cases of mass casualties (1, 2). It is possible that blast injury is under-reported due to the complicated multiple-injury scenario in which it is likely to occur. The use of enhanced blast weapons, particularly fuel-air-explosives (FAE), as well as the consequences of explosions in confined spaces, as in military operations in urban terrain and the blast inside armored fighting vehicles, poses an increased, though unquantitated, threat of blast injury. It is unlikely that blast injury would be the only wound sustained by a soldier. However, im-

proved field medical care, resuscitation, and transport, as well as wide use of personnel armor, may bring more casualties with serious blast injury to the field hospital.

The cloth ballistic vest (CBV) is a critical element of soldier protection from fragments and small-arms fire (3, 4). U.S. Army policy advocates its widest possible use in combat. Soldiers will wear it in intense blast environments. A previous study with volunteers exposed to low-level blast, equivalent to the overpressure routinely experienced by crew members around large-caliber artillery, has demonstrated a significant increase in intrathoracic pressure (ITP) when the CBV is worn compared to the ITP when only fatigues are worn (5). The peak ITP has been correlated with degree of injury and risk of mortality in high-level blast animal studies (6). This study was undertaken to evaluate the effect of the CBV on casualty level blast injury in an animal model.

METHODS

Sheep weighing an average of 38.2 kg (range, 29–53 kg) were sedated with intramuscular doses of nembutal. Individual sheep were suspended in a loose net sling with right side against the reflecting plate of a 3-meter diameter shock tube. Located on Kirtland AFB, New Mexico, the shock tube is 36.6 meters in length and open at one end. The explosive charge was a length of Primacord (328 grains/m) approximately 19 meters from the animal. Pressure measurements of the blast waves were made using piezoelectric transducers (Susquehana ST-4) mounted flush with the surface of the reflecting plate. Transducer signals were amplified and recorded on magnetic tape. Paper records

were obtained using a fiberoptic light beam oscilloscope, and descriptive parameters (peak pressure and duration) of the shock wave were read graphically. In a small number of animals, a small insulated piezoelectric pressure transducer (Atlantic Research LC-10) was placed trans-nasally into the thoracic esophagus 56 cm from the external nares. Intrathoracic pressure was recorded and analyzed as above.

Animals were selected in a blocked randomized fashion to be clad in a cloth ballistic vest (CBV) or to have no external garment. Those wearing the CBV were fitted in a size large U.S. Army-issue Kevlar* vest and the fastenings were adjusted to give a close fit. Blast levels were chosen to give a range of injury from threshold (no detectable lung weight change) to a level resulting in approximately 20% fatalities. Nominal levels of 115, 230, 295, and 420 kPa peak pressure were selected based on historical data (6). Following exposure to a single blast, the animals were transported to a laboratory where they were euthanized with a massive intravenous bolus of nembutal. A complete gross necropsy was performed within 1 hour of the shot. Particular attention was paid to the respiratory and gastrointestinal tracts as these organs have proven to be susceptible to blast injury (9, 11). All lesions were described and photographed. The lungs were removed in an inflated condition, trimmed of extraneous tissue, photographed, and weighed. Lung weights were expressed as a percentage of total body weight (LWI) (10, 11).

In conducting the research described in this report, the investigators adhered to the *Guide for the Care and Use of Laboratory Animals*, as promulgated by the Committee on Care and Use of Laboratory Animals of the Institute of Laboratory Animal Resources, National Research Council.

Data Analysis. All group values are reported as mean ± one standard deviation. A Duncan's *t*-test was performed on blast parameters and lung weight data on sets of animals exposed at the same nominal level with and without the CBV (7). A general linear methods ANOVA was performed on lung weight data. Mortality data were compared using Chi-square analysis. For all statistical tests, the level of significance was set at $p < 0.05$.

RESULTS

The weight of the animals wearing the CBV (38.94 ± 4.23 kg) was not different from those without (37.46 ± 4.05 kg). Table I summarizes the exposure condition and lung weight data. For the same nominal blast level, there was no difference in duration of the positive phase of the blast wave, peak pressure, or animal weight between groups with and without the CBV. Only at nominal 230 kPa was there a significant difference in lung weight between CBV and non-CBV animals ($p = 0.035$).

The values of lung weight are plotted against actual blast peak pressures in Figure 1. The trend towards higher lung weights in animals with the CBV is clear and is supported by the analysis of variance. Table II shows the expected strong correlation between lung weight and blast level in addition to a significant effect from use of the CBV. There was no demonstrable interaction between the blast level and CBV.

At the nominal 420 kPa level, two of six animals in the non-CBV group died compared to five of the six animals wearing the CBV. This results in a Chi-square of 3.09 for a 2 × 2 contingency ($0.05 < p < 0.10$). In a previous study without the CBV, at a similar blast level

TABLE I
Comparison of blast parameters and lung weight changes for matched sets of animals at the same overpressure levels with and without cloth ballistic vest (CBV)

Blast Peak (kPa)	Blast Duration (msec)	No.	CBV	Lung Weight % Body Weight
113 ± 5*	14.3 ± 0.2	6	No	0.94 ± 0.08
115 ± 4	14.4 ± 0.2	6	Yes	0.94 ± 0.06
232 ± 12	15.6 ± 0.7	9	No	1.22 ± 0.06
227 ± 4	15.4 ± 0.9	9	Yes	1.45 ± 0.26†
299 ± 26	15.2 ± 0.7	8	No	1.75 ± 0.57
294 ± 22	14.8 ± 1.2	8	Yes	2.17 ± 0.58
427 ± 20	13.6 ± 0.7	6	No	2.51 ± 0.39‡
417 ± 18	14.4 ± 1.1	6	Yes	2.62 ± 0.47§

* Data expressed as mean ± one standard deviation.

† Indicates significantly different ($p < 0.05$) from group at similar peak pressure without CBV.
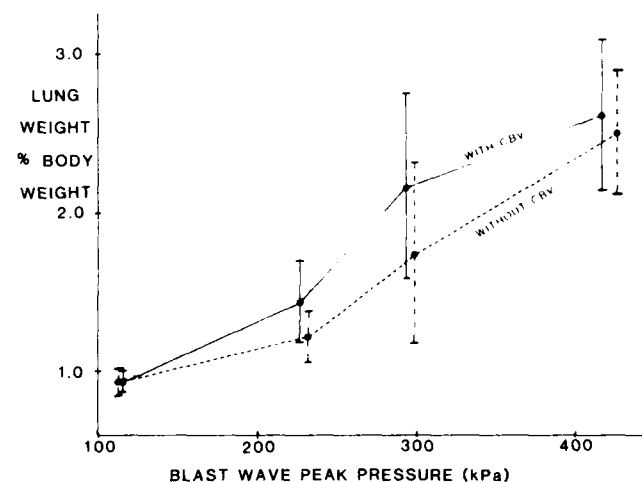
‡ Two animals died.

§ Five animals died.

FIG. 1. Comparison of effect of blast level on lung weight index in sheep with and without CBV. Data contained in Table I. * Groups are different $p < 0.05$.

TABLE II
Summary of analysis of variance for the effect on lung weight of use of the CBV, level of blast, and the interaction of blast level and CBV

Variable	DF	F value	Prob > F
Jacket	1	4.26	0.044
Blast level	2	44.7	0.0001
Jacket × level	2	0.79	0.503

only one of five animals died (15). If these mortality data are grouped, the CBV is associated with a statistically significant increase in mortality ($\chi^2 = 4.90$; $p < 0.05$).

Intrathoracic pressure (ITP) was measured in three animals with and three without the CBV at the 230 kPa level. For animals wearing the CBV the maximum ITP was 643 ± 241 kPa. For animals without the CBV the peak ITP was 516 ± 134 kPa. Because of the small

number of measurements, no statistical inferences are drawn.

DISCUSSION

Primary blast damage occurs almost exclusively in gas-containing structures, with pulmonary injury being the cause of immediate morbidity and mortality (9). Forces at air-tissue interfaces result in pleural, alveolar, and vascular disruption. Pneumothorax and pneumomediastinum may result. Diffuse hemorrhage occurs in lung parenchyma and leads to respiratory insufficiency. The use of post-exposure lung weight expressed as a percentage of total body weight (the lung weight index: LWI) is an accepted measure of pulmonary blast damage (9–11). Alveolar-venous fistulae allow air to enter the pulmonary veins and embolize to the systemic arterial circulation. Cerebral or coronary occlusion from air emboli is the major cause of early death.

Individual protection from primary blast effects has not proven to be practical. Clemedson and Jonsson demonstrated that a very strong, rigid covering would protect the thorax of small animals (10). The weight and bulk of such a system clearly makes it impractical for the individual soldier. In a companion study, the same investigators demonstrated a significant enhancement of lung injury when rabbits were wrapped in "protective" layers of foam rubber before blast exposure (10). Addition of layers of 50 and 200 mm of foam rubber resulted in a doubling of lung weights over the "unprotected" case and an increase in mortality. They also observed a large increase in peak internal pressure in a physical model of the rabbit when it was covered with foam. They attributed the worsening of injury to a damping of high frequency and enhancement of lower frequency components of thoracic vibration (10).

Previously, we carried out a study of the effects of various types of clothing and body armor on intrathoracic pressure (ITP) in volunteers (5). Subjects were exposed to levels of overpressure which were equivalent to that routinely experienced by the crews of large-caliber artillery (18.6 kPa peak). ITP was measured by a strain-gauge pressure transducer (Millar model PC-340) introduced transnasally into the esophagus and positioned 2 cm above the gastro-esophageal junction. Ten subjects were each clad in a random fashion in either fatigues alone or fatigues plus standard U.S. Army issue Kevlar® ballistic vest (2.9 kg), a military field jacket, a ceramic flak vest (6.4 kg), or the ceramic vest worn over the Kevlar. The maximum ITP in the Kevlar-only group (8.7 ± 1.2 kPa) was greater than in all other groups ($p < 0.05$ for difference from fatigues alone at 7.4 ± 0.7 kPa). No other clothing ensemble resulted in ITP measures which were different from that with fatigues.

The present study clearly shows that use of the CBV has a significant effect on pulmonary injury from blast (Fig. 1 and Tables I and II). The few measurements made

also suggest that ITP in animals at high blast conditions is increased by the wearing of the CBV. At the highest blast level studied, mortality was increased and may have resulted in an underestimation of the effect of the CBV. As the intensity of blast increases and animals die shortly after exposure, there is little time for hemorrhage to occur and the LWI may not reflect the severity of the injury (10, 11). This could explain the closeness of the mean LWI in the two groups at the 420 kPa level. It is likely that at an even higher blast level, the non-CBV group would have a higher mean LWI because of a disproportionate number of very early deaths (with consequent low LWI) in the CBV group. The ANOVA is also skewed by the data at 115 kPa. At this threshold level there should be no change in lung weight as the only pulmonary lesions were pleurally based petechiae in some animals. As anticipated, the CBV and non-CBV groups had identical mean LWI which is the same as historical control lung weight indices (9, 15). This also demonstrates that the jacket itself, irrespective of blast, had no effect on the lungs. However, it could affect the analysis of the data using a linear model. We expect that the CBV effect on lung weight change will be nonlinear and similar to that in Figure 1. At low levels there will be change in lung weight in either group and at mid-levels there will be a noticeable increase in LWI with the CBV. At some higher level the LWI's will again be similar due to the effect of early mortality on lung fluid accumulation as described above. However, removing either the 115 or the 420 kPa group from consideration did not have any major effect on the correlations shown in Table II.

At the 420 kPa level, five of six animals with the CBV died within 30 minutes as a result of the blast. Only two of the six animals without the CBV died. This blast intensity was chosen to give a 20% lethality in the non-CBV group (6, 11). Prior work at 420 kPa (15) had resulted in one of five animals dying. Combining the two groups without the CBV gives a mortality of 27% (three of 11). The 83% mortality in the CBV animals was significantly different ($p < 0.05$). The effect of increased mortality on LWI is discussed above. These two data points are shown in Figure 2, which is a log-probit plot (16). In order to estimate the mortality as a function of peak overpressure, straight lines were drawn through the data points using the common probit slope (5.593) determined for 13 species of animals (including sheep) exposed without the CBV to single blasts over a range of pressures and durations (6, 11). From Figure 2 it can be estimated that the use of a CBV reduces the overpressure necessary to give a given level of mortality by about 25%. For example, the lethal dose for 1% of the population (LD_1) is estimated to occur at 308 kPa for sheep without a CBV and 233 kPa for sheep with a CBV.

The mechanism of injury enhancement is unclear but its elucidation would add to our understanding of blast
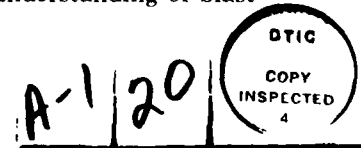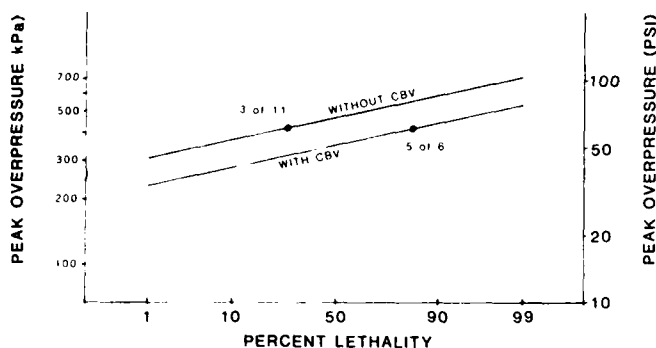
FIG. 2. Log probit plot for lethality following blast exposure. Mortality rates for 420 kPa group are plotted for animals with and without the CBV. Lethality lines are drawn using the common probit slope (5.593) determined for 13 species of animals (6, 11).

injury and pulmonary contusion in general. It is unlikely that the increase in the effective mass of the chest wall caused by donning the vest is significant. Modeling considerations suggest that such a change would be expected to decrease rather than raise the ITP at lower blast levels. The volunteer study failed to show greater effect for the circumstances of greatest mass, i.e., Kevlar and/ or ceramic vest. The increase in the surface exposed to the blast which accompanies the addition of a layer of material around a body may be an important factor. The force acting on a surface is the product of the pressure and the area affected. The larger "target" may collect more of the energy potentially available in the blast field and deposit it in the body. For example, a man with a chest circumference of 107 cm who is wrapped in a 3-cm covering will have his thoracic area, and hence force loading, increased by 18%. This may have been a factor in Clemedson's work where rabbits of about 100-mm diameter were wrapped in foam rubber 50 and 200 mm thick. Such coverings would increase the effective surface area by 100% and 400%, respectively. Such a bulky, compressible covering would be analogous to a lens, collecting energy from a large area, collapsing towards the animal and greatly increasing the load compared to the "unprotected" state. However, the volunteer study suggests that this is not the only factor. The much bulkier ceramic vest and ceramic-Kevlar combination did not increase ITP.

The mechanical coupling of the CBV through the intervening air and clothing to the body wall is an additional factor which may be critical. Measurements of the pressure under the CBV made during the volunteer study indicated that the sharp pressure rise of the free-field shock front had been converted to a load with a more gradual rate of application. If the CBV converted the loading function of the chest wall from the Friedlander function of the blast wave to one with a more gradual rise, then the transfer of energy to the lung might be more efficient. The relatively massive and stiff chest wall causes the thorax to have a low natural frequency on the order of 40 hertz (5, 11, 12). Thus the thorax cannot respond easily to the almost instantaneous shock front.

A loading function which takes place over a time period closer to the natural frequency of the system may result in higher chest wall velocities and hence a risk of greater injury (10). Measurements of chest wall motion and/or loads should be made in identical blast environments in animals with and without a CBV.

Blast injuries can be expected on the future battlefield. The use of a CBV may result in a greater incidence and severity of pulmonary blast injury. However, the risk of such injuries appears to be far less than the risk of wounding from fragments. The data in this report must not be construed as advocating that the CBV not be used. Rather it is to point out the risk of a particular kind of lung damage in modern combat. Given the proper setting, it is appropriate for physicians treating military casualties to consider the diagnosis of blast lung and the knowledge that a CBV was worn should heighten that suspicion. At present, guidelines for the treatment and triage of blast casualties are limited (13) and further research is necessary. Treatment strategies developed for blast injury can be applied to other causes of nonpenetrating chest trauma, including the contusion which occurs when a CBV stops a projectile (14).

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